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# The Croonian Lecture 2004 Risk: food, fact and fantasy

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We all take risks, but most of the time we do not notice them. We are generally bad at judging the risks we take, and in the end, for some of us, this will prove fatal. Eating, like everything else in life, is not risk free. Is that next mouthful pure pleasure, or will it give you food poisoning? Will it clog your arteries as well as filling your stomach? This lecture weaves together three strands—the public understanding of science, the perception of risk and the role of science in informing government policy—as it explains how food risks are assessed and managed by government and explores the boundaries between the responsibilities of the individual and the regulator. In doing so, it draws upon the science of risk assessment as well as our attitudes to risk in relation to issues such as bovine spongiform encephalopathy, dioxins in salmon and diet and obesity.

**Keywords:** food; risk; policy; BSE; dioxins; obesity

## 1. INTRODUCTION

As far as I can gather, this is the fourth Croonian Lecture, in its 266 year history, on 'food'—if you allow a bit of poetic licence. In 1740, Alexander Stuart's lecture was on peristaltic motion of the gut, which could loosely be taken as food talk. Over 200 years later, in 1955, C.H. Best talked about lipotropic agents, and a third 'food' lecture was given in 1963, by H.A. Krebs, on gluconeogenesis, a subject that I will touch on very briefly later. I am going to weave together three strands: the 'public understanding of science'; the perception of risk; and the role of science in informing government policy for managing risk.

When you hear people talk about the 'public understanding of science', you may think of the Royal Institution Christmas lectures, the writers of popular science books or the media scientists admired by the chattering classes. However, I am thinking of something less glamorous and more practical: how to present science-based policy to the public. Not long ago, there was a widespread view that if you explained the science more clearly, public worries about new technologies such as GM foods or nanotechnology would disappear. However, nowadays, the idea of 'the public' as an empty vessel waiting to be filled with scientific knowledge has been largely abandoned and replaced with the notion of a two-way conversation, in which the aim is to engage 'the public' and scientists in understanding each others' worries and perspectives. This approach is sometimes called 'Science and Society' (House of Lords 2000) or 'Science in Society' (Royal Society 2004). I will consider how this more recent approach is linked to the use of science to inform policy.

I will also discuss what happens when the scientific assessment of risk is out of tune with public perception, or when scientists disagree, or when scientists simply do not know. These are not rare or unusual events: far from it, they are closer to the norm. A related question is that of responsibility for managing risks, and whether different risks should be managed in different ways. (Many similar points are discussed from the perspective of an economist in Mervyn King's British Academy annual lecture 2004; King 2004).

## 2. EXPERTS ON RISK

Everyone has a view about food. During our adult lifetime, each of us in the UK will, on average, eat more than 80 000 meals amounting to some 60 million calories, or to put it another way, we each eat 600 chickens, 4000 loaves of bread and 5000 kg of potatoes. No wonder we all think of ourselves as food experts! We tend to think of ourselves as also being experts on risk. After all, we take calculated risks all the time, by crossing the road, driving the car, investing our money and so on. However, our judgement of risk may not be quite as good as many of us would like to think.

In a study conducted at Harvard Medical School, doctors were asked a question along the following lines (modified after Eddy 1982; see also Gigerenzer 2002):

> Suppose you suspect a patient has cancer, you send them for screening, and the results come back positive. You know that the test isn't perfectly accurate: it detects cancer, if cancer is there, in 80% of patients. You also know that the cancer in question affects about 1% of patients and that the test gives a false positive result on about 10% of tests. The test result comes back positive. What should you tell your patient about their risk of having cancer?

On average, doctors say that the risk is 75%. The correct answer is 7.5%. Think of it like this: one in every 100 patients has cancer. Of these, the test detects 0.8 but the test also indicates cancer for 9.9 of the 99 patients who do not have the disease (the false

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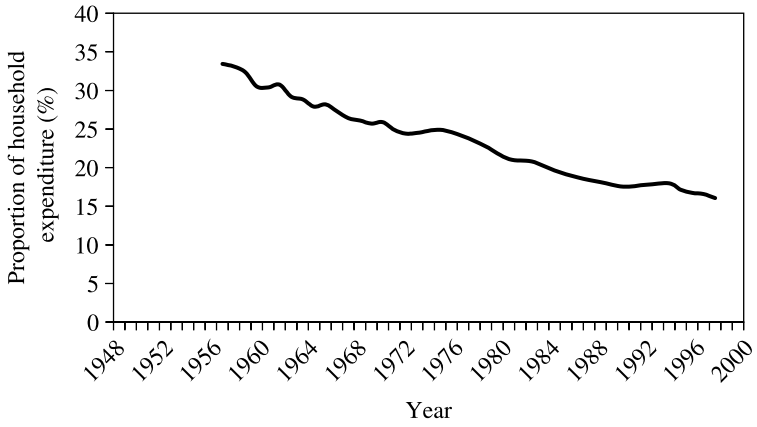


Figure 1. Food is getting cheaper, in relative terms (based on household expenditure on food and soft drinks from the Family Expenditure Survey; Pascoe 2004, unpublished work).

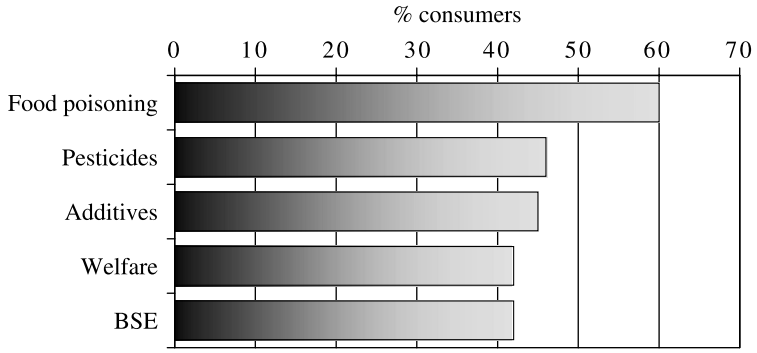


Figure 2. Proportion of consumers expressing concern about specific food issues, from a list of 14 food issues (Food Standards Agency 2004*b*).

Table 1. How many deaths create a news story? (Harrabin *et al.* 2003.)

| issue | deaths per story |
|---|---|
| smoking | 8571 |
| obesity | 7500 |
| alcohol | 4724 |
| mental health | 1222 |
| vCJD | 0.33 |
| measles | 0.25 |

positives). So the risk of having the disease, given a positive test result, is $0.8/(0.8+9.9)\times 100=7.5\%$.

## 3. FOOD RISKS

What about food risks? For the majority in the UK, our food lives are probably better than ever before. Food is not only cheaper (figure 1) and more varied than it was for our parents and grandparents, it is probably safer now than it ever was in the past. For instance, in 1938, although it was known that over 2500 people a year died from drinking raw milk in Britain, the risk was not seen as big enough to justify legislation to make pasteurization compulsory (Pennington 2003).

We now expect much higher standards of food safety, and unlike the 800 million people worldwide who risk starvation every year (Food and Agriculture Organisation 2003), we can enjoy the luxury of fear of relatively minor risks. However, if you look in the newspapers, you might sometimes think that we are continually confronted with serious food risks. Of course, much of this is drama rather than news. It is true that there is, on average, at least one food safety incident a day (Food Standards Agency 2004*a*), but the vast majority of these are trivial. If you ask people to rank food risks from a prompt list, the top five are usually food poisoning, pesticides, additives, welfare and bovine spongiform encephalopathy (BSE) (figure 2). The answers are, of course, restricted to the items on the list.

The media write stories that people want to read, so they mirror and amplify peoples' concerns not just about food, but about health scares in general. A survey by the King's Fund has shown, for example, that a death from variant CJD (an unbelievably harrowing and tragic loss of young life) attracts, in proportion, 23 000 times the media coverage of a death from obesity (table 1).

What about the scientific evidence for food risks? One crude measure of risk is the number of premature deaths per year attributed to a food-related cause. It is crude because death is only one measure of risk and because death may be a result of multiple causes, the relative importance of each being difficult to disentangle. Accepting these limitations, this very approximate scale shows strikingly that the big food risks we all face are the dietary contributions to cardiovascular disease and cancer (table 2). Health economists sometimes express relative risks as 'disability adjusted life years' (DALYs), a method that takes into account morbidity, mortality and age at which the risk affects people. Recalculating the risks in table 2 as DALYs would alter

Table 2. Food risks: UK deaths per year related to diet or food.
(Assuming around a third of cancer and coronary heart disease deaths are diet-related.)

| risk | food deaths/year |
|---|---|
| cancer | 56 000[a,b] |
| coronary heart disease | 35 000[a,c] |
| food-borne illness | *ca* 500[d] |
| vCJD | <20[e] |
| food allergy | *ca* 10 |
| GMOs, pesticides, growth hormones | 0 |
| choking on food | 150[f] |

[a] Department of Health (2001).
[b] Royal College of Physicians of London (2004).
[c] British Heart Foundation (2001).
[d] Adak *et al.* 2002.
[e] Department of Health (2004*a*).
[f] Office for National Statistics 2002.

the absolute numbers but not their order of magnitude. Since chronic diseases such as cardiovascular disease and cancer tend to be diseases of older people, their contribution would be somewhat lower when expressed in DALYs (Jamison 2004, personal communication).

With a conservative epidemiological estimate that about one-third of the risk for cancer and coronary heart disease are diet-related, between them they are responsible for more than 90 000 premature diet-related deaths per year. To this must be added a proportion of the 170 000 deaths from stroke, for which diet is an acknowledged, but not yet quantified, contributor. One estimate for the USA is that there are 500 000 diet-related premature deaths per year, which scales approximately with population size.

In comparison, each year in the UK about 500 people die prematurely of food-borne illness (Adak *et al.* 2002), and so far, fewer than 30 people from vCJD in the year with the largest number of cases (Department of Health 2004*a*). Each of these deaths is tragic at the individual level, but at the population level these food risks are relatively small. There are no reported deaths from pesticides, GMOs or veterinary medicines in food. To give a context for these risks, choking to death on food apparently causes about 150 deaths per year, and getting in or out of bed about 100 deaths (Office for National Statistics 2002). So one message is, if you want a risk-free life do not eat or sleep!

## 4. PERCEPTION OF RISK

Is there an explanation for the difference between actual (table 2) and perceived (figure 2) risk? Psychologists such as Paul Slovic (Slovic 2000) have suggested one possible account. They have shown that risks with certain characteristics, often summarized as 'unknown' and 'dread', are perceived as being big and those with opposite characteristics are seen as small (figure 3). 'Unknown' means new, not observable, delayed in effect and not well understood. 'Dread' means uncontrollable, involuntary, inequitable and potentially catastrophic. For a summary of a wide range of research in this area, including discussion of many kinds of bias in the perception of risk, see Kahnemann & Tversky (2000).

Thus, given that the way people see risks may differ radically from the scientific assessment of their magnitude, should regulators concentrate on managing the risks that are actually big, or those that are thought to be big? It is tempting to answer by saying that people are irrational in their appreciation of risk. No objective analysis of risk would say it is safer to ride a bike down a busy street than to live near a nuclear power station, but many people would chose the first rather than the second.

Another way to look at it is that intuitive perceptions are in some way justified. Risks that are unknown and potentially catastrophic should be given more weight than the simple statistics suggest, especially when there is no identified benefit; and benefit is an important part of the equation of public perception of risk. For instance, the public response to the use of genetic modification in medicines is very different to that for food (Gaskell *et al.* 2000; Gaskell & Bauer 2001; Poortinga & Pidgeon 2003), probably because the medicines bring benefits while, at least for the moment, the foods do not.

My conclusion is that we should rely on science to assess risks, recognizing that science may not have crisp certainties in its answers. Scientific knowledge has a status that sets it apart from perception, belief and hunch. However, there may not be a simple, linear translation of the assessment of risk into policy for managing risk, and here perception of risk, or in other words, its acceptability, might come into play.

## 5. MANAGING RISK

Translating science into policy involves judgements about what is acceptable to the public, the degrees of uncertainty and the costs and benefits. These judgements, sometimes dignified with the title of the 'precautionary principle' (European Commission 2004*a*), cannot be captured in a formula, but one way of quantifying them is to calculate the implied value placed on a human life as a result of spending money to manage risks. The more the government spends to save a life, the more precautionary it is.

There are two ways of looking at this. One is to ask what people think should be spent, and the other is to calculate what is actually spent by government as a result of current regulations.

### (a) *Willingness to pay*

Asking people what they are willing to pay (WTP) can be done directly, for instance, through questionnaires/interviews, or indirectly, through studying what people earn as a premium for doing risky jobs Neither is by any means perfect. The direct method has the problem of assuming that people understand the question (phrased in statistical terms rather than for the individual or her family) and give a fair answer. (For a fuller discussion of different approaches to estimating WTP see Jones-Lee *et al.* 1995 and Beattie *et al.* 1998.) The indirect method assumes that people taking riskier jobs understand the risks and that other factors do not influence the premium (Viscusi & Gayer 2002). In spite of these

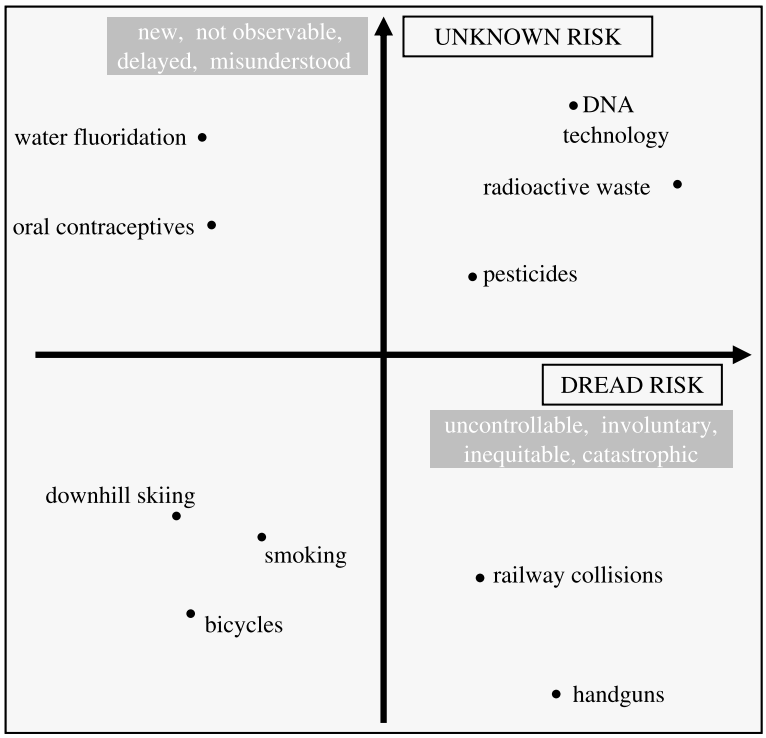


Figure 3. Risks can be mapped in relation to their sense of 'dread' and the degree to which they are 'unknown'. Risks that score high on these two axes are perceived as bigger than risks that score low (after Slovic 2000).

Table 3. Implicit value per life: the actual amounts spent to save a life ($1995) based on the cost of a selection of US health and safety regulations.
(Viscusi & Gayer 2002.)

| regulation | cost per life saved ($m) |
|---|---|
| seat belt/airbag | 0.1 |
| car side-impact standards | 1.0 |
| asbestos | 24.7 |
| formaldehyde | 256 373 |

potential problems, studies in the USA arrive at similar statistical 'values per fatality' (VPF) from the two approaches—approximately US$6 million (Viscusi & Gayer 2002). The comparable figure most frequently quoted for the UK is £1.25 million (Department for Transport 2003).

It would not be surprising if people were WTP more to save lives when the risks have high 'dread' or are 'unknown', but where this has been studied, the differences are surprisingly small and not always in the expected direction. For instance, in Britain, people say they are WTP about 20% more per life for road than for rail safety, even though rail transport has a higher dread score. Even after the Ladbroke Grove crash, in which 31 people died, the VPF for rail was only about 15% higher than for road when regular train users were asked, and similar to road for infrequent train users (Chilton *et al.* 2002).

### (b) *Implicit value per life*

The actual amounts spent to save a life, reflecting political judgements about risk management, vary enormously, from a few thousand to many tens of millions or even billions of dollars for different risks. Table 3 shows data for the USA, but the UK would be broadly comparable. Why the huge variation? Some regulations, such as restrictions on environmental pollution, are not just about protecting human life, but are also about broader concerns for society. Furthermore, political decisions about risk management and regulation are often influenced by particular disasters: the Ronan Point disaster in the case of tall buildings, the Ladbroke Grove rail crash in the case of rail transport. Immediately after the crash, ministers proposed fitting automatic train protection (ATP) to all trains, with an implicit value per life of £15 million. Following the recommendations of an independent enquiry, the decision was taken to install the cheaper train protection and warning system (TPWS). This still implies a VPF of £7 million (Chilton *et al.* 2002), considerably higher than the £1.25 million referred to earlier.

### (c) *Who should manage risks?*

By no means are all risks managed by regulation Often, and rightly, people are left to make up their own minds. Also, businesses manage risks as part of their provision of products and services and to protect their reputations. Is there consistency about who is expected to manage risk? Should it be government, individuals, industry?

For risks that are perceived as high, people are more likely to want the government to step in. On the other hand, for risks that are seen as low, people expect to be left to choose for themselves. For instance, GM foods, perceived to be risky, but with no known adverse effect on human health, are tightly regulated, while salt, sugar and the saturated fat content of processed foods, all of which can contribute to well-documented and substantial risks, are not. Finally, it is worth a reminder that, although I am talking about risks in isolation, benefits come into the equation too: all judgements

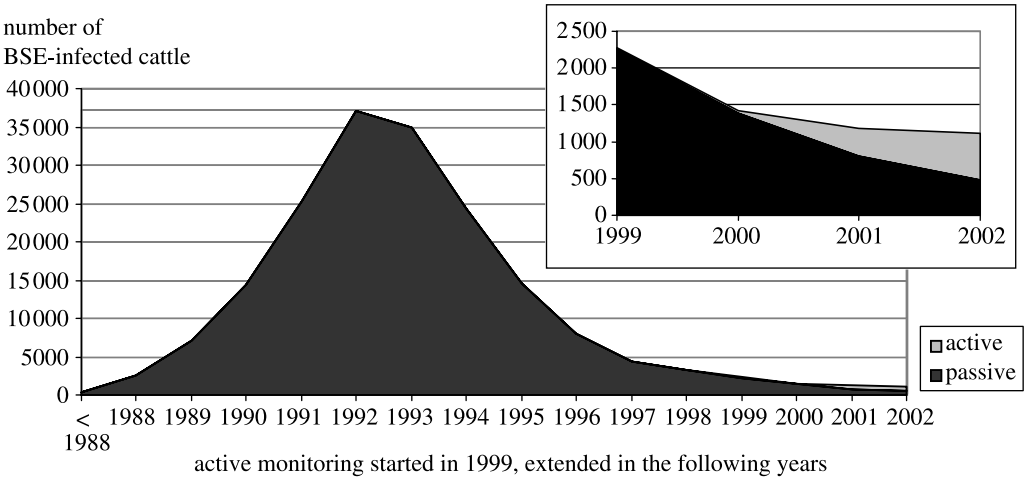


Figure 4. BSE cases in the UK detected by active monitoring and passive surveillance. Active monitoring refers to testing of animals for BSE; passive surveillance means the observed clinical cases (European Commission 2002, unpublished work.).

about risk imply a judgement about the balance of risks and benefits.

Let me summarize so far. People do not necessarily see risks in the same way as the scientific assessment. When it comes to managing risks, judgements are made about how precautionary to be. These may depend in part on what the science says, in part on what people think, and in part on what politicians think, as will be illustrated in the following examples.

## 6. BOVINE SPONGIFORM ENCEPHALOPATHY

Bovine Spongiform Encephalopathy is the most significant event in the recent history of the management of food risk and in the role of science in informing policy. Its legacy is still with us. As the *Guardian* leader of 10 March 2004 put it: 'Successive governments have too often relied on the imprimateur of science to win support for controversial policy ends, or simply to avoid embarrassment. The examples of BSE and human variant CJD are still fresh in the collective memory'. I do not have time to go into the history of BSE, other than to note that one lesson from this event for scientists, was to police the boundary between their job of risk assessment and the political job of risk management.

### (a) *BSE uncertainties*

We now know more about BSE (and related diseases) than we did a decade ago, but there are still many unknowns, including why it first arose, how it is transmitted, the extent of the species barrier, how it spreads within the body and the relationship between different variants of the disease Although the prion hypothesis is widely accepted, there is still some dispute about the nature of the agent that causes the disease.

The link between BSE and variant Creutzfeld–Jakob Disease (vCJD) is of course circumstantial, although the two diseases appear to be caused by the same abnormal prion and have a similar characteristic pathology, as well as being associated in their epidemiology. Some of the more recent observations appear to increase, rather than decrease the uncertainty about BSE: the possibility of blood transfusion as a route of infection for vCJD (Hansard 2003), the possibility that some cattle may have a form of BSE closer to sporadic CJD (Casalone *et al.* 2004) and the appearance of BSE in North America (US Department of Agriculture 2004).

Additional uncertainty about the eventual size of the human vCJD epidemic has been raised as a result of a retrospective survey of about 12 000 tonsils and appendices, screening for a histological staining pattern indicative of vCJD (Hilton *et al.* 2004). The prevalence estimates from this survey could be taken to indicate a higher level of infection in the population than is apparent from the clinical cases to date, although the sample size of 'positives' is very small (three out of approximately 12 000), with only one of the three possible positives considered clear cut.

### (b) *The UK BSE epidemic*

In spite of these uncertainties, it is clear that the BSE epidemic in the UK has declined rapidly in the past few years (figure 4). The number of new clinical cases each year is now a trickle—in 2003 it was approximately 180—compared with 37 000 at its peak in the early 1990s. This is because what is generally regarded as the main route of transmission, feed containing remains of animals, was cut off progressively, with a fully effective ban since mid-1996. At the same time, the number of deaths from variant CJD, considered to be the human form of BSE, each one a tragic loss, has risen more slowly than epidemiologists originally estimated. There have been 153 UK cases up to the end of 2004, and 148 deaths (figure 5). The modellers are saying that a few thousand deaths in total is a very pessimistic estimate, with a few hundred being most likely. This must be hedged with caution; there could be multiple waves of the disease, although the most parsimonious hypothesis would predict that the first wave would be the largest. Furthermore, as mentioned earlier, the estimate of the total epidemic from the cases to date is incompatible with the most pessimistic estimate from the tonsil and appendix survey.

### (c) *BSE risk management*

There are two main planks of the current risk management policy to protect people from vCJD. Importantly,

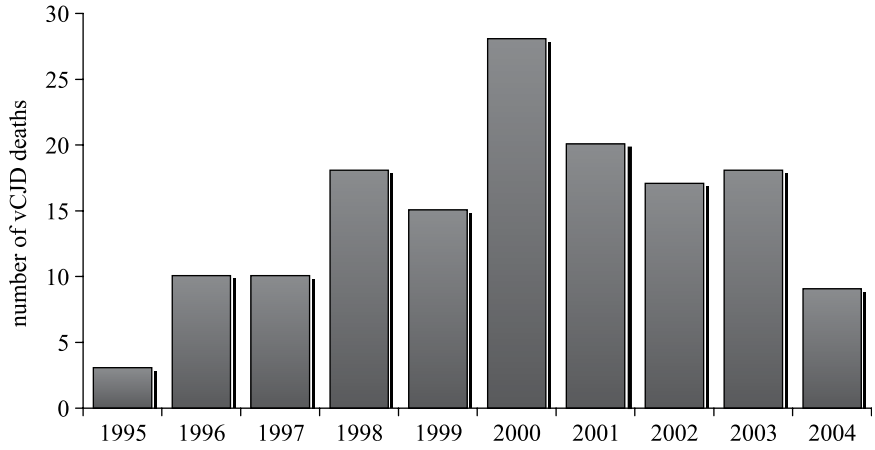


Figure 5. Deaths (definite and probable) from vCJD in the UK, 1995–2004 (Department of Health 2004*a*).

Table 4. BSE in Europe—tests on apparently healthy animals (2001–2003).
(European Commission 2004*b*.)

| country | number tested (million) | positive cases from tests | total BSE |
|---|---|---|---|
| France | 8.20 | 194 | 907 |
| Germany | 7.67 | 101 | 305 |
| Spain | 1.24 | 146 | 412 |
| EU total | 26.21 | 840 | 5601 |

neither of these should be seen as aiming for zero risk. In the memorable words of Lord Phillips, government's role is to 'reduce risk to a level acceptable to the reasonable consumer' (Phillips 2000). This leaves the terms 'reasonable' and 'acceptable' open to interpretation.

The two measures are these: first, the bits of the body most likely to contain infectivity (e.g. brain, spinal cord, some internal organs) are kept out of the food chain (the so-called specified risk material, SRM, controls); second, animals more than 30 months old are killed and destroyed instead of going for food (Food Standards Agency 2000). The SRM controls are estimated to remove more than 99% of the infectivity from an animal with BSE. The rationale behind the 'over thirty month rule' (OTM) is that the disease takes about 5 years to develop to clinical symptoms, and that by 30 months, even an infected animal will contain relatively little infectivity. Thirty months is an arbitrary cut off, because obviously the development of the disease is continuous.

Other countries in Europe that have had much smaller BSE epidemics (table 4) do not have an OTM rule. Instead, animals are tested, and if they are BSE-negative they go into the food chain. The tests are known to be reliable for animals with clinical symptoms or within a few months of showing symptoms, but not for early stages of the disease (Moynagh *et al.* 1999).

### (d) *The switch to testing*

Although the UK epidemic was far larger than in any other European country, the incidence of BSE in more recent years is not (figure 6), and the government has, on the basis of a risk assessment commissioned by the Food Standards Agency (FSA), decided to switch to the European-wide system of testing animals over the age of 30 months. This has been a judgement on how precautionary to be or to put it the other way round, a judgement on how much it is worth spending to achieve a particular level of protection.

How will the risk change as a consequence of a change to testing? There could be a tiny increase in risk—because the test does not always pick up pre-clinical cases of BSE. In the most pessimistic realistic assumption, this extra risk was estimated to be of the order of 0.5 extra vCJD deaths over the next 60 years, as the result of exposure over the next 5 years (Food Standards Agency 2004*c*).

Three things should be said about this estimate. First, the number should not be taken literally. There are so many assumptions and uncertainties, that it means 'a very small extra risk indeed, perhaps zero, perhaps a few extra deaths'. (For an excellent brief discussion of inappropriate use of complex models in estimating risk, see May (2004).) In order to handle this uncertainty, the extra risk was expressed as a fraction of the risk already there, which allows many of the uncertainties to cancel out. In other words, assuming that, as a result of past exposure, between 5000 and 20 000 people are already fatally infected, the extra number in the future from a switch to testing is probably between zero and two.

Second, the estimate nevertheless depends on a number of assumptions, for instance, the total number of vCJD cases, the efficacy of the test, and so on. In estimating the risk, the experts used a range of values from 'best guess' to 'pessimistic'. This gives an approximation of the uncertainties but is not a guarantee that the assumptions are correct. The calculations assumed that the tonsil and appendix result, rather than the cases to date, reflect the true potential scale of the vCJD epidemic. If the cases to date are taken as a more appropriate basis for the risk assessment, the estimates of additional risk would be much smaller.

Third, the assessment was fully discussed in public so that all interested parties could challenge it and see

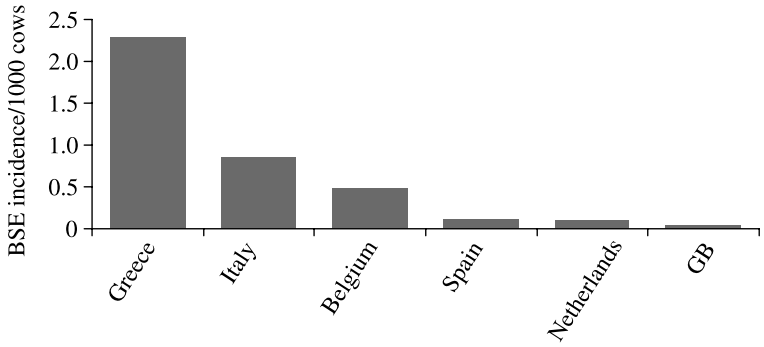


Figure 6. BSE incidence in Europe per 1000 cows born in 1997 (Donnelly *et al.* 2002).

the assumptions and uncertainties that went into it. Transparency and independence from vested interests are central prerequisites for public confidence in scientific assessment of risk (Lewontin 2004).

The political judgement about whether or not the small (perhaps zero) additional risk, together with the uncertainties, were sufficient to retain the OTM scheme rather than switch to testing included both a consideration of public acceptability and confidence, and an estimate of the degree of precautionary protection. Bovine Spongiform Encephalopathy is so iconic in terms of public confidence and trust in risk management, as well as having many features of 'dread', that a very precautionary stance is justified. However, if you take the best estimate of about half a life saved over 60 years as the result of the next 5 years of the OTM scheme, the implied value of preventing a fatality is about £2 billion. Recognizing that the assessment of risk still has uncertainties, the judgement is that a managed transition to testing is the appropriate choice.

### (e) *BSE and sheep*

In spite of all its uncertainties, BSE risk from cattle is well characterized when you compare it with the possible risk of BSE in sheep. The correct answer to the question, 'Is there BSE in sheep?' is 'We do not know; there is a possibility that it is there at a low level'. BSE could have spread into sheep at the same time as it spread in cattle because sheep ate the same feed and they can develop BSE in the laboratory by eating infected material. No BSE has been found in the commercial flock, but it could be there at a low level, masked by a related disease, scrapie.

Scrapie has been endemic in our sheep since the early eighteenth century and does not seem to transmit to humans. Some sheep with scrapie have been tested to see if they actually have BSE, and so far all the results have been negative (Food Standards Agency 2003). Approximately 200 have been tested with the most reliable method, injecting infected material into mice to see if they develop BSE or scrapie. About another 2000 have been tested with a biochemical test, which is currently being validated in a European ring trial. This testing reduces the uncertainty, but cannot prove the absence of BSE, only that, if it is there, its prevalence is very low.

Here is a risk whose magnitude is unknown, and may be zero. Should it be managed by, for example, banning sheep meat? The political judgement is 'no', that would not be proportionate. There is some precautionary risk management—akin to the SRM controls for cattle referred to earlier—but these are known to be only partially effective in reducing the possible risk. However, telling people about uncertainty can help them to manage their own risks by choice, as well as building confidence by being honest. For instance, in 2001, when the FSA made a public statement about the possibility of BSE in sheep (Food Standards Agency 2001*a*), the Meat and Livestock Commission (2001) measured the public response. Although a surprising number (66%) of people were aware of the issue, most decided not to change their eating habits (Meat and Livestock Commission 2001).

## 7. DIOXINS AND SALMON

My BSE examples illustrated where science ends and political judgement starts, and the importance of being honest in telling people what the scientists do not know. But what about when the experts disagree? For practitioners of science, disagreement among experts is familiar. The landscape of scientific understanding has areas where facts are relatively secure, like substantial mountain ranges, and others regions where knowledge is shifting and ephemeral, like quicksand. We would all be more than a little shocked if we woke up one morning to find that some scientists were claiming that the genetic material was not DNA or RNA, but we would hardly raise an eyebrow if a new theory of consciousness appeared. In between, in the great mass of scientific knowledge, there is a majority view at any one time, and there are dissenters. However, there are different kinds of dissenters. Some are visionary individuals who against the odds turn out to be right, while others are wrong, misguided or even perhaps making a living out of the role. Then there are groups who harness dissenters' results for their own purposes to pursue a political agenda (reminding us of James McNeill Whistler's comment: 'I maintain that two and two would continue to make four, in spite of the whine of the amateur for three, or the cry of the critic for five').

People love reading stories about heroes and villains, and it is easy to paint the minority scientist as the plucky, downtrodden hero fighting the attempts of the powerful scientific establishment to gag her. We can all think of examples in the past few years: the MMR vaccine, GM potatoes and BSE caused by organophosphates. (For further reading, see Liess & Powell's *Mad*

*cows and mother's milk* (1998).) However, the story of dioxins and salmon is not just about an individual dissenter, but about disagreement among official regulators. Dioxin-like compounds (of which there are more than 200, including dioxin-like polychlorinated biphenyls (PCBs)) are by-products of past industrial activity and they are rapidly declining in the environment, so that people in Britain are exposed to less that half the level of eight years ago. Some of them cause cancer in animal tests. The evidence for a similar effect in humans mainly comes from industrial accidents or occupational exposure that has resulted in very high doses. Although some of these indicate an increase in some cancers, the results are not totally clear-cut. There is also some suggestion that some compounds could have adverse effects on the cognitive and reproductive development of children, and this is still under investigation. (For a good overview see Food Standards Agency (2001*b*) or Institute of Medicine (2003).)

### (a) *Scientific disagreement*

Early in 2004, a paper in *Science* reported that dioxin levels (or more accurately, dioxin-like compounds) in farmed salmon, especially Scottish salmon, were so high that people should not eat more than two or three servings a year (Hites *et al.* 2004). The consumer, trying to figure out what was going on, heard two rival accounts: one was from the authors of the paper, reputable experts publishing in one of the top scientific journals, and the other from food safety or health authorities such as the Food Standards Agency (2004*d*), the Food and Drug Administration (see ABC News 2004) and the World Health Organization (WHO 2004). These authorities all agreed that the data did not vary from previous results on dioxin levels in salmon and that these levels did not cause concern for people eating salmon once a week (considerably above the current average consumption). What is more, there are health benefits from eating oily fishes such as salmon, so people should carry on as they are: the benefits outweigh the small risks.

Why did the two sets of experts, one a majority, the other a minority, disagree? The authors of the *Science* paper (who are, incidentally, an example of scientists straying across the boundary from risk assessment to policy) followed the risk assessment methodology of the US Environmental Protection Agency (EPA). The EPA assumes that dioxins can cause cancer at any dose, however low (EPA 2000). If you take the EPA position as a general guide for your diet, it would be difficult to eat a balanced diet because of the ubiquity of dioxins in food. The other authorities refer to the available evidence on how dioxins act to cause cancer (Food Standards Agency 2001*b*; JECFA 2001; SCF 2001). They do not act directly on DNA but rather by binding to a receptor site. This leads to the conclusion that, at very low levels, dioxins have essentially no adverse effect, because compensatory mechanisms allow normal cell functioning (figure 7).

Thus there is a centre of gravity of scientific opinion, but a spread of views around this centre. This is not unusual. A survey that asked toxicologists to agree or disagree with a set of simple propositions showed that, while the professionals agree on many things, such as the dose-dependence of toxic effects, there is a fairly even split over some basic issues. These included the extent to which animal studies can be used to estimate human toxicity, and the extent of as yet unknown risks from toxic chemicals (Slovic 2000).

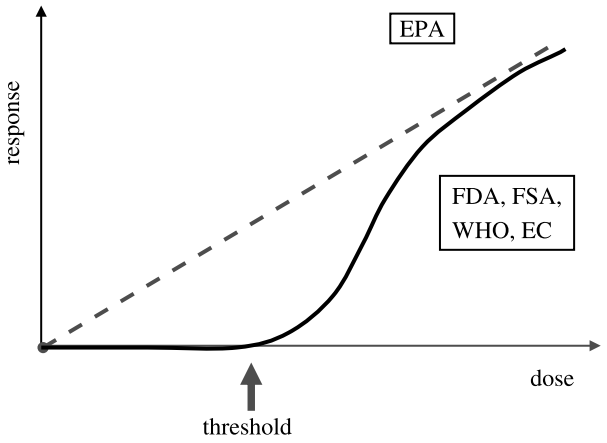


Figure 7. Dioxin risk assessment. The US Environment Protection Agency (EPA) assumes a linear dose–response relationship, whereas the US Food and Drug Administration (FDA), World Health Organization (WHO), Food Standards Agency (FSA) and European Commission (EC) assume a threshold below which there is no effect. See text for further explanation.

What is the consumer to make of this? The FSA's focus group work at the time showed that few people were spontaneously aware of the whole salmon affair. When it was explained, the typical reaction was cynicism about scientists scaremongering, mixed with a view that it was American scientists stirring up trouble for Scottish salmon farmers! The lesson for scientists is to find more effective ways of conveying the fact that scientific opinion is usually not monolithic, and that this does not undermine the validity of scientific advice.

Finally, before leaving the salmon story, it is worth adding that, as with worries about GM food, environmental concerns were (perhaps deliberately) mingled with food safety. Many of the reports dwelt on the adverse environmental impacts of salmon farming and on the ecological inefficiency of raising top predators (the marine equivalent of lions) as food animals. Since people in general are not as interested in the environment as in their own health, a health scare story is more effective than one about environmental damage. The study's claim that farmed salmon have higher dioxin levels than wild salmon was confounded by the fact that the wild salmon came from the Pacific and the farmed from the Atlantic. The FSA data comparing farmed and wild Atlantic salmon showed no difference and showed higher levels in herring than in salmon.

## 8. DIET AND HEALTH

The biggest food risks we all face in Western society are the ones that are not normally even considered as 'risks' in the traditional sense. They are the risks that result from eating too much of the wrong kinds of

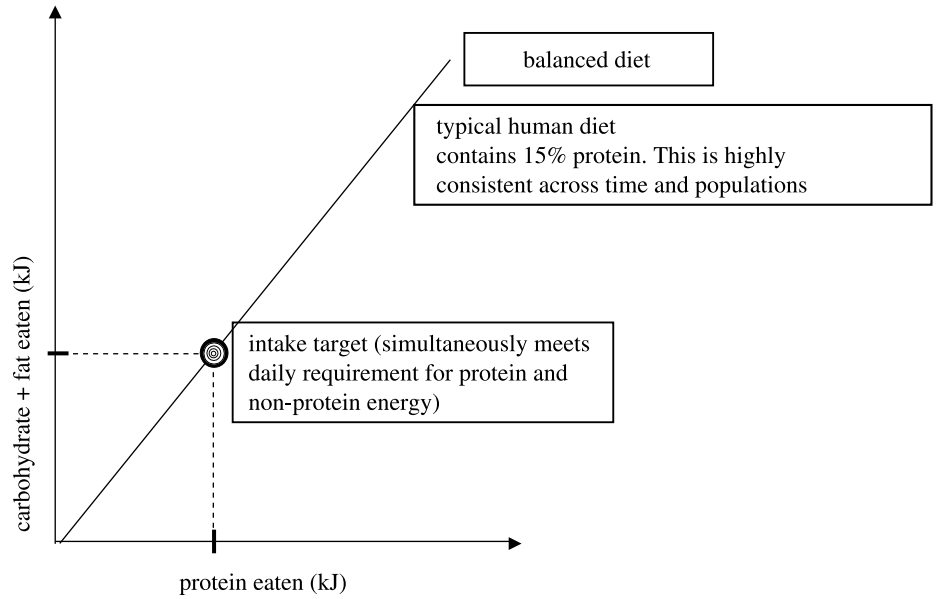


Figure 8. Protein leverage hypothesis (I): a balanced diet where 15% of energy is provided by protein. The body's homeostatic mechanisms lead to a balance in energy consumption at 15% protein and a daily intake along the diagonal line will meet this balance. A typical intake target is shown (Simpson *et al.* 2003).

foods. The facts are well-known. As a population we eat too much salt (Scientific Advisory Committee on Nutrition 2003), which contributes to high blood pressure and thence cardiovascular disease. We also eat too much saturated fat and not enough fruit, vegetables and fibre.[1] Children face the same problems, with likely health consequences for their future, but this risk is seen as voluntary, under our own control. Diet is a 'lifestyle choice', so most people assume that individuals or parents should take responsibility for their management. This contrasts sharply with, for instance, the much smaller BSE risks I talked about earlier.

### (a) *Promotion of food to children*

If you argue that diet is about information and choice, you have to ask: what information, what choice? Children are bombarded with information about foods that are high in fat, sugar and salt, just the things they ought to eat less of. Some people say it is obvious that this influences children's diets, while the food industry has said that it does not, it only affects brand loyalty.

The FSA's conclusion (Hastings *et al.* 2003), which fits with common sense and parental experience, is that advertising does influence children's diets. It influences categories (chocolates versus crisps) and not just brands (Kit-Kat versus Mars), and the quantitative effect cannot be disentangled from other influences such as parents and peers, because all of these interact. The evidence consists of a mixture of correlative studies and experiments in which children are exposed to TV adverts and then offered choices. No one study is definitive and the evidence is not black and white. After the FSA's review was completed, more than one industry-sponsored academic came out with critiques and counter-arguments (Paliwoda & Crawford 2003; Young 2003), but when the various analyses were put before an independent group of experts, the group supported the FSA review's conclusions.

In the Government's recent White Paper (Department of Health 2004*b*), the evidence from the FSA review is used as the basis for suggesting steps for tightening up the voluntary restrictions on advertising food to children, but the final, political, decision on what and how should be tightened up has yet to be made.

### (b) *Diet and obesity*

Although some people talk of obesity and diet as though they were the same problem, this is incorrect. Dietary health extends more widely than obesity, and obesity is influenced by both what you eat and how much you exercise—both sloth and greed. In the USA, caloric intake has gone up over the past 20 years (Cutler *et al.* 2003), while in the UK, the survey data suggest that it has not (Henderson *et al.* 2003*b*), although it now seems that people, especially those who are obese, under-report what they eat by about 30% (Rennie *et al.* 2005). Meanwhile, indicators of energy expenditure suggest that sloth has gone up (Royal College of Physicians of London 2004), although most people who want to lose weight do so by dieting. A recent analysis by the Health Development Agency shows that losing weight by exercise alone is less easy than by dieting or by a combination of diet and exercise (Health Development Agency 2003).

Thus, at a simple level, the exhortation must be to eat fewer calories and expend more. But why do people eat too many calories in relation to their expenditure? Why does the body's homeostatic mechanisms not keep intake in balance with expenditure? Some say we are fooled by extra large portion sizes (Rolls 2003). Others say that processed food with a high energy density and little bulk fools the body's short-term satiation mechanisms that depend

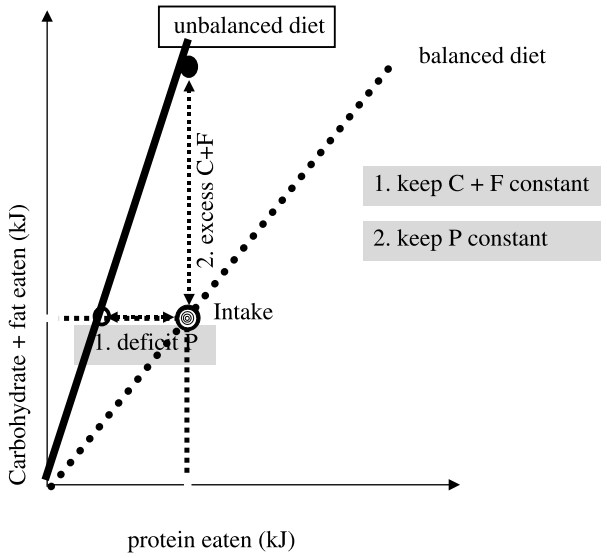


Figure 9. Protein leverage hypothesis (II): if the proportion of energy from protein in an individual's diet is low, below the 15% 'set point' (solid line), a substantial excess of energy (from carbohydrate+fat) is consumed to compensate for the protein deficit. An increase in the proportion of energy provided by protein is required to restore an energy balanced diet (dotted line; Simpson *et al.* 2003).

in part on stomach distention (Prentice & Jebb 2004*a*). Still others argue that the great variety of food available may make it difficult to stop eating (Rayner & Epstein 2001). What is more, in our evolutionary history, fat and sugar were important necessities for survival, so we are tuned to like eating them and they are now superabundant (Prentice 2001; Drewnowski & Levine 2003; Levine *et al.* 2003). Perhaps, in addition, the homeostasis of intake cannot cope with very low levels of expenditure (Prentice & Jebb 2004*b*). These ideas are not mutually incompatible and all of them, as well as others, may have some validity.

### (c) *The protein leverage hypothesis*

Simpson *et al.* (2003) have recently suggested an ingenious explanation of how dietary imbalance could lead to obesity. It is a current idea and still needs to be thoroughly evaluated. Their basic tenet, supported by evidence, is that, of the macronutrients, protein is more tightly regulated than fat and carbohydrate. For instance, across different countries, there is much less variation in protein intake than there is in fat and carbohydrate intake. If the diet contains only a percentage or two less protein than the body's 'set point' of 15%, people will eat substantially more fat and carbohydrate in an attempt to regulate protein to this level (figures 8 and 9).

If Simpson *et al.* are right, one contribution to obesity may be the low protein content of a diet of processed food. Typically, this is less than 10%, and, in aiming for the 15% target, individuals might increase their consumption of fat and carbohydrate by as much as 40%. In the USA, for instance, over the past 40 years the average diet has dropped from 14 to 12.5% protein. In order to maintain protein intake in the face of this decline, people have to eat 14% more fat and carbohydrate. However, the hypothesis does not end there. Simpson *et al.* also suggest that there is a positive feedback loop. Obese people, they hypothesize, have increased levels of free fatty acids in the blood, which in turn inhibits the insulin control of gluconeogenesis from amino acids in the liver. This results in protein depletion, and hence a further urge to increase food intake to restore protein levels.

The link between diet and exercise could also operate through this mechanism. Lack of exercise is associated with increased insulin resistance and hence enhanced gluconeogenesis. Protein leverage could also explain how high protein diets, such as the Atkins diet, could, in theory, work. Crucially, it would be necessary only to increase protein in the diet to the level that matches the set point. This is not meant to be an endorsement of Atkins: there are dangers in eating high levels of protein and cutting back too much on complex carbohydrates. The Simpson *et al.* hypothesis, which is still in its early stages of development, has potentially wide implications for policy in managing obesity, but balance is still the boring king.

## 9. CONCLUSIONS

When it comes to risk, the notion of 'one size fits all'—that there is a set level of acceptable risk for all aspects of our lives—is wrong. Acceptable risk varies across kinds of risk, according to how unknown they are and how much they are involuntary and uncontrollable. People also implicitly balance risks against benefits. So new technologies with immediate benefits, such as mobile phones, are embraced by the population without much concern for possible risk, while GM foods, which currently bring no benefits to consumers, are not.

Science is crucial for the assessment of risk, but all too often, there is incomplete knowledge or disagreement among the experts. This is the reality. For policy-makers it means that judgements about risk management have to be made in the light of uncertainty, sometimes called the precautionary approach. For scientists it means more straightforward acknowledgement of the strengths and limitations of their evidence.

Finally, we should not assume that increasing scientific literacy, while it may help, will alter public reaction. Frank Zappa referred to pop music journalism as 'People who can't write, interviewing people who can't talk for people who can't read'. We certainly should aim to avoid such tri-partite illiteracy in science education, but at the same time we should recognize that a scientifically literate public may well be a more sceptical one.

I thank Roger Lakin for his extensive help in preparing the lecture and Belinda Wood for checking references.

## ENDNOTE

[1]The National Diet and Nutrition Survey (various references supplied below: Gregory *et al.* 2000; Henderson *et al.* 2002*a*,*b*; Henderson *et al.* 2003*a*,*b*; Rennie *et al.* 2005) is the most comprehensive source of data on what people actually eat.

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